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Article

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Non-communicating extradural arachnoid cyst: a rare case report with review of literature

Asheesh Kumar Gupta, Anand Sharma, Avdhesh Shukla, S.N. Iyengar

Department of Neurosurgery, G.R. Medical College Gwalior, INDIA

Abstract: Extradural arachnoid cysts in the spine are uncommon causes of spinal cord compression in the paediatric population that are thought to arise from congenital defects in the duramater. In most literatures it is describe that such cysts communicating with the intrathecal subarachnoid space through a small defect in the dura. In this case report we describe a case of a child who presented with spinal cord compression caused by a large spinal extradural arachnoid cyst that did not communicate with the intradural subarachnoid space. An 9 year-old girl presented with progressive lower-extremity weakness, myelopathy, and severe gait ataxia. Magnetic resonance imaging of the spine demonstrated a large extradural arachnoid cyst extending from T4 to T9. The patient underwent a thoracic laminectomy for en bloc resection of the spinal extradural arachnoid cyst. Intra-operatively, the dura was intact and there was no evidence of communication into the intradural subarachnoid space. Postoperatively, the patient's motor strength and ambulation improved immediately, and no subsequent cerebrospinal fluid leak occurred.

Key words: EAC (Extradural arachnoid cysts), CSF (Cerebro spinal fluid)

Introduction

Extradural arachnoid cyst is a rare entity accounting for 1% of all spinal tumors (1). Pathogenesis remains unclear and thought to be congenital or develop secondary to infection or trauma. In nearly all cases of EAC, communication of CSF between the cyst and the intrathecal subarachnoid space through a dural defect has been reported.

In this report we present the case of a 9 year-old female with a symptomatic extradural spinal arachnoid cyst of dorsal spine treated successfully by surgery. This case is unique because there was no dural defect or fistulous communication into the intradural subarachnoid space.

Case report

A 9 year old female presented with history of back pain followed by difficulties in walking. Neurological examination revealed spasmodic paraparesis. On neurological examination, she had 4/5 strength in all muscle groups of lower limb. Proprioception and

vibratory sensation were diminished in both legs. Her reflexes were hyperactive and her gait was severely ataxic. She had no cutaneous stigmata of neurological disease. Muscle tone at the lower extremities was increased. MRI spine showed a large extradural cyst located posterior to the cord from T4 to T9 with dorsal compression and flattening of thecal sac.

The mass was hypointense on T1-weight imaging, was hyperintense on T2 weight weighted imaging, and suppressed signal intensity on fluid-attenuated inversion recovery imaging.

The signaling patterns were consistent with CSF suggestive of a spinal extradural arachnoid cyst. No obvious communications between the cyst and the intrathecal subarachnoid space were detected on neuroimaging.

Management

After discussion of risks and benefits, patient underwent T4 to T9 laminectomy; lamina overlying the cyst was thinned. A large extradural cyst resembling an arachnoid cyst was identified, with a thick but translucent wall. The cyst was easily dissected from the exterior surface of the thecal sac. The dura mater was inspected and there was no evidence of a dural defect, arachnoid pedicle, or fistulous communication into intradural sub arachnoid space.

Histopathological finding were consistent with arachnoid cyst. The patient's motor function improved to full strength and she was ambulating well. CSF leak did not develop, and he was discharged on the 10th postoperative day.

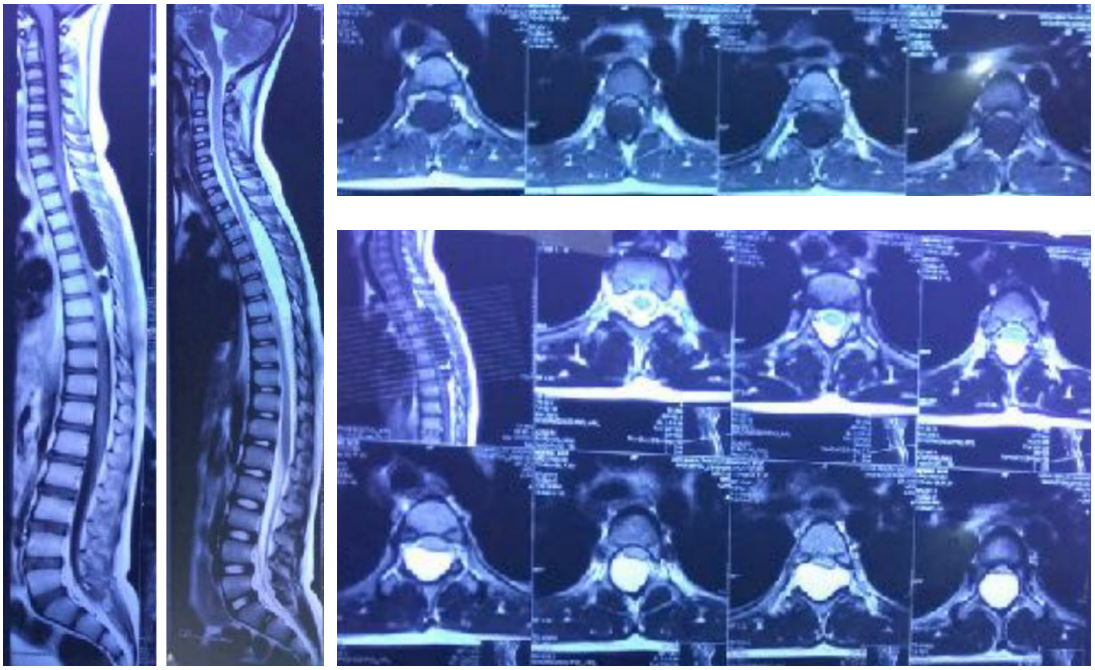

Figure 1- MRI Spine saggital and axial view with T1 and T2 sequences showing extradural arachnoid cyst compressing dural tube

Figure 2 - Showing excised extradural arachnid cyst

Discussion

Nabors et al. (2) described a classification system of spinal arachnoid cysts divided into three major categories: extradural cysts without spinal nerve root fibers (Type I); extradural cysts with spinal nerve root fibers (Type II); and intradural cysts (Type III). Type I is further divided into extradural arachnoid cysts (Type IA) and sacral meningoceles (Type IB). In nearly all cases of Type IA cysts, communication of CSF between the cyst and the intrathecal subarachnoid space through a dural defect has been reported. Our case represents the type IA variant.

The exact origin and pathogenesis of Type IA spinal extradural arachnoid cysts remain unknown. It is an out-pouching herniation of arachnoid membrane through a dural defect that may communicate to intradural subarachnoid space (3). The etiology of this herniation is still unclear and can be either congenital or acquired. (4) In 1934, Elsberg, et al.(5), attributed the origin of these cysts to either congenital diverticula of the dura or herniation of arachnoid membrane through a congenital dural defect. Congenital dural defects appear most commonly at the dural sleeve of the nerve root or at the junction of the sleeve and the thecal sac; they derive less commonly from the dorsal midline of the thecal sac. Although less common, spinal arachnoid cysts have been associated with arachnoiditis, surgery, and trauma, leading others to suggest that these cysts arise from acquired dural defects (6).

Spiegelmann, et al.(7), reported a case in which hemosiderin-containing macrophages found in the cyst wall in a patient with a spinal extradural arachnoid cyst caused spastic paraparesis 10 years after craniospinal injury suggesting acquired etiology for SEAC.

Mechanisms of growth for these cysts are not well known. It may be pulsatile CSF dynamics, ball-flap mechanism or osmotic gradient (8). According to the theory of McCrum and Williams, (9) intermittent surges of pressure in the subarachnoid space are communicated to the cyst and flow occurs into the pouch. When pressure falls again, egress of fluid is impeded by compression of the cyst pedicle. According to the Laplace law, the body of the cyst exerts a force on the neck sufficient to close the communication, because its radius and wall tension are greater. This mechanism then allows further enlargement, with persistent CSF pulsations. Bone erosion of the spinal canal may imply the presence of a valve mechanism that is responsible for producing forces of CSF pressure within the

cyst that are greater than normal hydrostatic forces (10).

Our case is unusual in that there was no detectable dural defect or communication with the intradural subarachnoid space. We postulate that the noncommunicating cyst evolved from a preexisting communicating cyst that initially formed as a result of a small dural defect secondary to spinal trauma. Over time, the cyst enlarged and eventually obliterated the communicating channel to the subarachnoid space because of the Laplace law (1). The closure of the dural defect may be due to the proliferation of arachnoid cells. Computerised tomography myelography is the diagnostic study of choice to demonstrate the communication of the cyst with the subarachnoid space (1) however, this study was not performed in our case.

Correspondence
Dr. Asheesh Kumar Gupta M.Ch. resident
Department of Neurosurgery G.R.
Medical College Gwalior.
Address- Room no. 39 Senior Boys Hostel
G.R. Medical College Gwalior
Mobile No.- 7828832632
Email- asheesh_gsvm@yahoo.com

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